CLAIMS

1. An *in vivo* assay to measure phenotypic stability of a certain cell population (i.e. the capacity to form stable cartilage *in vivo* by isolated chondrocytes) comprising subcutaneous or intramuscular injection in a mammal of a cell suspension (i.e. articular chondrocytes) in an iso-osmotic liquid, the same suspension comprising articular chondrocytes in an amount equivalent to at least 1×10^6 chondrocytes as applied to immune-deficient mice.
2. Use of the *in vivo* assay of claim 1 to evaluate the possibility that a treatment administered to a certain cell population can hamper or enhance the anchorage-independent growth of said population as well as its phenotypic stability (e.g. the cartilage forming ability of a chondrocyte suspension).
3. Use of the *in vivo* assay of claim 1 to optimize cell culture conditions and manufacturing processes for a specific application in tissue engineering (i.e. chondrocyte expansion for autologous chondrocyte transplantation).
4. Use of the *in vivo* assay of claim 1 to predict the outcome of autologous cell transplantation using a certain population of cells placed in a certain differentiation pathway as a means to predict phenotypic stability.
5. Use of the *in vivo* assay of claim 1 to identify molecular markers linked to the phenotypic stability of a certain cell population in a certain differentiation pathway.
6. A method of identification of molecular markers linked to the outcome of the *in vivo* assay of claim 1, comprising using freshly isolated or serially passaged cells from a certain cell population placed in a certain differentiation pathway using also differential gene expression analysis methods including differential display, subtractive hybridization, subtracted libraries or cDNA chips and cDNA arrays.
7. Use of BMP-2 and/or FGFR-3 and/or markers co-detecable with these markers and/or specific reporter constructs or molecules belonging to the specific intracellular signalling pathways as molecular markers positively associated with chondrocyte phenotypic stability.
8. Use of activin-like kinase-1 (ALK-1) and/or markers co-detectable with these markers and/or specific reporter constructs or molecules belonging to the specific intracellular signalling pathways as molecular markers negatively associated with chondrocyte phenotypic stability.
9. Use of sets of positive and/or negative markers for chondrocyte stability provided on DNA arrays or DNA chips for routine detection of chondrocyte stability .
10. Use of sets of positive markers for chondrocyte stability wherein the positive marker

is present in phenotypically stable primary chondrocytes and chondrocytes, after at least one passage, that remained phenotypically stable.

11. Use of molecular markers of phenotype stability identified in claim 5 or claim 6 or defined in claims 7 or 8 as tools to monitor passage by passage cell expansion and/or to predict when cell expansion must be stopped and/or to recover cells that have already lost their phenotypic stability only when needed and/or to provide a means for quality control of cells to be used for autologous cell transplantation.
12. Use of molecular markers positively associated with phenotypic stability nd/or molecular markers negatively associated with phenotypic stability as identified in claims 5, 6, 7 or 8 for selecting from a cell population only those cells that retain their phenotypic stability.
13. Method of cell sorting via antibodies in accordance with claim 11 or 12 wherein the antibodies are monoclonal or polyclonal antibodies.
14. Method of cell sorting via antibodies according to claims 11 or 12 wherein the antibodies are raised against FGFR-3 and/or ALK-1.
15. Method of cell sorting via antibodies according to claims 11 to 13 wherein the antibodies raised against FGFR-3 are raised against a fragment of FGFR-3.
16. Method of cell sorting via antibodies according to claim 15, wherein the antibodies are raised against an epitope of the extracellular domain of FGRF-3 or against the synthetic peptide TGLVPSERVLVGPQRLQVLNASHEDSGAYSCRQRLTQRVL.
17. Method of cell sorting via antibodies according to claim 16 wherein the epitope is comprised between the I and the II Ig-like domain.
18. Polyclonal antibody specifically recognizing part of the extracellular domain of FGFR-3 and as such useful for cell sorting, obtainable by immunizing animals with FGFR-3 or with the synthetic peptide TGLVPSERVLVGPQRLQVLNASHEDSGAYSCRQRLTQRVL.
19. Polyclonal antibody specifically recognizing part of the extracellular domain of FGFR-3 and as such useful for cell sorting, obtained by immunizing animals with FGFR-3 or with a fragment therof or with the synthetic peptide TGLVPSERVLVGPQRLQVLNASHEDSGAYSCRQRLTQRVL.
20. Selection method using the antibodies of claims 16 to 19, comprising FACS, sorting via protein-conjugated magnetic beads, affinity chromatography or any other suitable means of sorting based on antibodies directed against cell surface markers.
21. Use of cells retaining their phenotypic stability and selected according to claim 12 for transplantation to a connective tissue site in a patient according to claim 12 for transplantation to a connective tissue site in a patient or for seeding any prosthetic

device intended to be anchored into a mammal host.

22. A therapeutical composition including cells selected according to any of the claims 11 to 16.

23. A therapeutical composition according to claim 22, further including at least a pharmaceutically acceptable carrier and/or a growth factor.

24. A diagnostic comprising the DNA chips of claim 9.

25. A diagnostic comprising at least one of the antibodies of claims 18 or 19 for quality control of chondrocytes.

26. A diagnostic comprising antibodies raised against any of the products of the genes of the DNA chips of claim 9 for quality control on chondrocytes.

27. A cell culture exhibiting chondrocyte phenotypic stability.in which the cells express a ratio of BMP-2 and/or FGFR-3 and/or markers co-detecable with these markers and/or specific reporter constructs or molecules belonging to the specific intracellular signalling pathways as molecular markers positively associated with chondrocyte phenotypic stability to activin-like kinase-1 (ALK-1) and/or markers co-detectable with this marker and/or specific reporter constructs or molecules belonging to the specific intracellular signalling pathways as molecular markers negatively associated with chondrocyte phenotypic stability which is greater than 1, preferably greater than 2.

28. A cell culture exhibiting chondrocyte phenotypic stability in which the cells do not express activin-like kinase-1 (ALK-1) and/or markers co-detectable with this marker and/or specific reporter constructs or molecules belonging to the specific intracellular signalling pathways as molecular markers negatively associated with chondrocyte phenotypic stability.